File 155:MEDLINE(R) 1950-2006/Aug 31
(c) format only 2006 Dialog
File 55:Biosis Previews(R) 1993-2006/Aug W4
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File 34:SciSearch(R) Cited Ref Sci 1990-2006/Aug W4
(c) 2006 The Thomson Corp
File 434:SciSearch(R) Cited Ref Sci 1974-1989/Dec
(c) 2006 The Thomson Corp
File 340:CLAIMS(R)/US Patent 1950-06/Aug 31
(c) 2006 IFI/CLAIMS(R)
***File 340: IPCR/8 classification codes now searchable in 2006 records.**
For important information about IC=index changes, see HELP NEWSIPCR.

```
      Set  Items  Description
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? s apoptosome
      S1     988  APOPTOSOME
? s caspase(w)9
           74197  CASPASE
         5311095  9
      S2   10332  CASPASE(W)9
? s s1 and s2
             988  S1
           10332  S2
      S3     478  S1 AND S2
? s review
      S4 1192839  REVIEW
? s s3 and s4
             478  S3
         1192839  S4
      S5      21  S3 AND S4
? rd

>>>Duplicate detection is not supported for File 340.

>>>Records from unsupported files will be retained in the RD set.
      S6      16  RD  (unique items)
? t s6/3,k,ab/1-10
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6/3,K,AB/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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20924851 PMID: 16678772
The apoptosome : **physiological, developmental, and pathological modes of regulation.**
Schafer Zachary T; Kornbluth Sally
Department of Pharmacology and Cancer Biology, Duke University Medical Center, Durham, North Carolina 27710, USA.
Developmental cell (United States) May 2006, 10 (5) p549-61, ISSN 1534-5807--Print Journal Code: 101120028
Contract/Grant No.: R01 CA102702; CA; NCI
Publishing Model Print
Document type: Journal Article; Review
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Apoptosis, a form of programmed cell death, is executed by a family of zymogenic proteases known as caspases, which cleave an array of

intracellular substrates in the dying cell. Many proapoptotic stimuli trigger cytochrome c release from mitochondria, promoting the formation of a complex between Apaf-1 and **caspase - 9** in a caspase-activating structure known as the **apoptosome** . In this **review** , we describe knockout and knockin studies of **apoptosome** components, elegant structural and biochemical experiments, and analyses of the **apoptosome** in various cancers and other disease states, all of which have provided new insight into this critical locus of apoptotic control.

The apoptosome : **physiological, developmental, and pathological modes of regulation.**

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6/3,K,AB/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

15140094 PMID: 15505412
Apoptosome **dysfunction in human cancer.**
Hajra K M; Liu J R
Department of Obstetrics and Gynecology, University of Michigan Medical School, L4000 Women's Hospital, 1500 East Medical Center Drive, Ann Arbor, MI 48109, USA.
Apoptosis - an international journal on programmed cell death (United States) Nov 2004, 9 (6) p691-704, ISSN 1360-8185--Print
Journal Code: 9712129
Publishing Model Print
Document type: Journal Article; Review
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Apoptosis is a cell suicide mechanism that enables organisms to control cell number and eliminate cells that threaten survival. The apoptotic cascade can be triggered through two major pathways. Extracellular signals such as members of the tumor necrosis factor (TNF) family can activate the receptor-mediated extrinsic pathway. Alternatively, stress signals such as DNA damage, hypoxia, and loss of survival signals may trigger the mitochondrial intrinsic pathway. In the latter, mitochondrial damage results in cytochrome c release and formation of the **apoptosome** , a multimeric protein complex containing Apaf-1, cytochrome c , and **caspase - 9** . Once bound to the **apoptosome** , **caspase - 9** is activated, and subsequently triggers a cascade of effector caspase activation and proteolysis, leading to apoptotic cell death. Recent efforts have led to the identification of multiple factors that modulate **apoptosome** formation and function. Alterations in the expression and/or function of these factors may contribute to the pathogenesis of cancer and resistance of tumor cells to chemotherapy or radiation. In this **review** we discuss how disruption of normal **apoptosome** formation and function may lead or contribute to tumor development and progression.

Apoptosome **dysfunction in human cancer.**

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...the pathogenesis of cancer and resistance of tumor cells to chemotherapy or radiation. In this **review** we discuss how disruption of normal **apoptosome** formation and function may lead or contribute to tumor development and progression.

6/3,K,AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14692814 PMID: 14699955
The role of neurotransmission and the Chopper domain in p75 neurotrophin receptor death signaling.
Coulson E J; Reid K; Shipham K M; Morley S; Kilpatrick T J; Bartlett P F
Queensland Brain Institute, University of Queensland, Brisbane, Qld, Australia. e.coulson@uq.edu.au
Progress in brain research (Netherlands) 2004, 146 p41-62, ISSN 0079-6123--Print Journal Code: 0376441
Publishing Model Print
Document type: Journal Article; Review
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
The role of p75 neurotrophin receptor (p75NTR) in mediating cell death is now well characterized, however, it is only recently that details of the death signaling pathway have become clearer. This **review** focuses on the importance of the juxtamembrane Chopper domain region of p75NTR in this process. Evidence supporting the involvement of K+ efflux, the **apoptosome** (**caspase - 9** , apoptosis activating factor-1, APAF-1, and Bcl-xL), caspase-3, c-jun kinase, and p53 in the p75NTR cell death pathway is discussed and regulatory roles for the p75NTR ectodomain and death domain are proposed. The role of synaptic activity is also discussed, in particular the importance of neutrotransmitter-activated K+ channels acting as the gatekeepers of cell survival decisions during development and in neurodegenerative conditions.

... it is only recently that details of the death signaling pathway have become clearer. This **review** focuses on the importance of the juxtamembrane Chopper domain region of p75NTR in this process. Evidence supporting the involvement of K+ efflux, the **apoptosome** (**caspase - 9** , apoptosis activating factor-1, APAF-1, and Bcl-xL), caspase-3, c-jun kinase, and...

6/3,K,AB/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14538198 PMID: 14563117

The CD95 type I/type II model.

Barnhart Bryan C; Alappat Elizabeth C; Peter Marcus E
The Ben May Institutefor Cancer Research, University of Chicago, 924 E. 57th Street, Chicago, IL 60637, USA.

Seminars in immunology (United States) Jun 2003, 15 (3) p185-93, ISSN 1044-5323--Print Journal Code: 9009458
Contract/Grant No.: GM61712; GM; NIGMS
Publishing Model Print
Document type: Journal Article; Review
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

CD95 (APO-1/Fas) has become the prototype of a death domain containing receptor and is the best studied member of the death receptors that activate the extrinsic apoptosis pathway. This pathway is initiated by recruitment and activation of caspase-8, an initiator caspase, in the death-inducing signaling complex (DISC) followed by direct cleavage of downstream effector caspases. In contrast, the intrinsic apoptosis pathway starts from within the cell either by direct activation of caspases or through intracellular changes such as DNA damage resulting in the release of a number of pro-apoptotic factors from the intermembrane space of mitochondria. The release of these factors results in the activation of another initiator caspase, **caspase - 9** , and ultimately in the activation of effector caspases in a protein complex called the **apoptosome** . In recent years, it has become apparent that there is cross talk between the extrinsic and intrinsic pathway. In the death receptor pathway of apoptosis induction, the best characterized connection between the two pathways is the Bcl-2 family member Bid which translocates to mitochondria after cleavage by caspase-8 causing pro-apoptotic changes. Cells that die through CD95 without help from mitochondria are called Type I cells, whereas cells in which CD95-mediated death relies mostly on the intrinsic pathway are called Type II. This **review** focuses on recent developments in the delineation of the biochemistry and the physiological function of the two CD95 pathways.

... of mitochondria. The release of these factors results in the activation of another initiator caspase, **caspase - 9** , and ultimately in the activation of effector caspases in a protein complex called the **apoptosome** . In recent years, it has become apparent that there is cross talk between the extrinsic...

... which CD95-mediated death relies mostly on the intrinsic pathway are called Type II. This **review** focuses on recent developments in the delineation of the biochemistry and the physiological function of...

6/3,K,AB/5 (Item 1 from file: 55)

DIALOG(R)File 55:Biosis Previews(R)

0015328158 BIOSIS NO.: 200510022658

Regulation mechanism of selective atresia in porcine follicles: Regulation of granulosa cell apoptosis during atresia

AUTHOR: Manabe Noboru (Reprint); Goto Yasufumi; Matsuda-Minehata Fuko; Inoue Naoko; Maeda Akihisa; Sakamaki Kazuhiro; Miyano Takashi
AUTHOR ADDRESS: Univ Tokyo, Anim Resource Sci Ctr, Res Unit Anim Life Sci, Ibaraki 3190206, Japan**Japan
AUTHOR E-MAIL ADDRESS: amanabe@mail.ecc.u-tokyo.ac.jp
JOURNAL: Journal of Reproduction and Development 50 (5): p493-514 OCT 04

2004
ISSN: 0916-8818
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: More than 99% of follicles undergo a degenerative process known as "atresia", in mammalian ovaries, and only a few follicles ovulate during ovarian follicular development. We have investigated the molecular mechanism of selective follicular atresia in mammalian ovaries, and have reported that follicular selection dominantly depends on granulosa cell apoptosis. However, we have little knowledge of the molecular mechanisms that control apoptotic cell death in granulosa cells during follicle selection. To date, at least five cell death ligand-receptor systems [tumor necrosis factor (TNF)alpha and receptors, Fas (also called APO-1/CD95) ligand and receptors, TNF-related apoptosis-inducing ligand (TRAIL; also called APO-2) and receptors, APO-3 ligand and receptors, and PFG-5 ligand and receptors] have been reported in granulosa cells of porcine ovaries. Some cell death ligand-receptor systems have "decoy" receptors, which act as inhibitors of cell death ligand-induced apoptosis in granulosa cells. Moreover, we showed that the porcine granulosa cell is a type II apoptotic cell, which has the mitochondrion-dependent apoptosis-signaling pathway. Briefly, the cell death receptor-mediated apoptosis signaling pathway in granulosa cells has been suggested to be as follows. (1) A cell death ligand binds to the extracellular domain of a cell death receptor, which contains an intracellular death domain (DD). (2) The intracellular DD of the cell death receptor interacts with the DD of the adaptor protein (Fas-associated death domain: FADD) through a homophilic DD interaction. (3) FADD activates an initiator caspase (procaspase-8; also called FLICE), which is a bipartite molecule, containing an N-terminal death effector domain (DED) and a C-terminal DD. (4) Procaspase-8 begins auto-proteolytic cleavage and activation. (5) The auto-activated caspase-8 cleaves Bid protein. (6) The truncated Bid releases cytochrome cfrom mitochondrion. (7) Cytochrome c and ATP-dependent oligimerization of apoptotic protease-activating factor-1 (Apaf-1) allows recruitment of procaspase-9 into the **apoptosome** complex. Activation of procaspase-9 is mediated by means of a conformational change. (8) The activated **caspase - 9** cleaves downstream effector caspases (caspase-3). (9) Finally, apoptosis is induced. Recently, we found two intracellular inhibitor proteins [cellular FLICE-like inhibitory protein short form (cFLIPs) and long form (cFLIPL)], which were strongly expressed in granulosa cells, and they may act as anti-apoptotic /survival factors. Further in vivo and in vitro studies will elucidate the largely unknown molecular mechanisms, e. g. which cell death ligand-receptor system is the dominant factor controling the granulosa cell apoptosis of selective follicular atresia in mammalian ovaries. If we could elucidate the molecular mechanism of granulosa cell apoptosis (follicular selection), we could accurately diagnose the healthy ovulating follicles and precisely evaluate the oocyte quality. We hope that the mechanism will be clarified and lead to an integrated understanding of the regulation mechanism.

...ABSTRACT: of apoptotic protease-activating factor-1 (Apaf-1) allows recruitment of procaspase-9 into the **apoptosome** complex. Activation of procaspase-9 is mediated by means of a conformational change. (8) The activated **caspase - 9** cleaves downstream effector caspases (caspase-3). (9) Finally, apoptosis is induced. Recently, we found two...

DESCRIPTORS:

MISCELLANEOUS TERMS: ...Literature **Review**

6/3,K,AB/6 (Item 2 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)

0014879132 BIOSIS NO.: 200400249889
Regulation of apoptosis proteins in cancer cells by ubiquitin.
AUTHOR: Zhang Huang-Ge; Wang Jianhua; Yang Xinwen; Hsu Hui-Chen; Mountz John D (Reprint)
AUTHOR ADDRESS: Department of Medicine, Division of Clinical Immunology and Rheumatology, The University of Alabama at Birmingham, 701 South 19th Street, LHRB 473, Birmingham, AL, 35294, USA**USA
AUTHOR E-MAIL ADDRESS: john.mountz@ccc.uab.edu
JOURNAL: Oncogene 23 (11): p2009-2015 March 15, 2004 2004
MEDIUM: print
ISSN: 0950-9232 _(ISSN print)
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Ubiquitin inhibitors act at many levels to enhance apoptosis signaling. For TNF-related apoptosis-inducing ligand (TRAIL)-mediated apoptosis signaling, there are at least five mechanisms by which apoptosis are regulated by the ubiquitin-proteasome pathway. First, proteasome inhibitors can decrease Fas-like inhibitor protein (FLIP) protein levels in tumors, resulting in increased apoptosis signaling due to increased caspase-8 activation. This appears to involve the ubiquitin ligase TNF receptor activation factor-2 (TRAF2) and acts indirectly by causing cell-cycle arrest at a stage where there is high degradation of the FLIP-TRAF2 complex. Second, the regulation of the proapoptotic Bcl-2 family member BAX occurs indirectly. Apoptosis signaling and caspase activation results in a confirmation change in the normally monomeric BAX, which exposes the BH3 domain of BAX, leading to dimerization and resistance to ubiquitin degradation. BAX then translocates into the mitochondria, resulting in the release of proapoptotic mitochondrial factors such as cytochrome c and second mitochondria-derived activator of caspase (SMAC). This results in the activation of **caspase - 9** and formation of the **apoptosome** and efficient apoptosis signaling. A third mechanism of the regulation of TRAIL signaling in the ubiquitin-proteasome pathway is mediated by the inhibitor of apoptosis proteins (IAP) E3 ligases. These IAPs can directly bind to caspases but also can act as ubiquitin ligases for caspases, resulting in the degradation of these caspases. IAP binding to caspases can be inhibited by SMAC, which exhibits a **caspase - 9** homology domain. The fourth mechanism for apoptosis activation by proteasome inhibitors is through the stabilization of the inhibitor of the kappaB (IkappaB)/NF-kappaB complex and prevention of nuclear translocation of the antiapoptosis transcription factor NF-kappaB. During TRAIL-DR4, DR5 signaling, this pathway is activated by interactions of activated Fas-associated death domain with activated receptor-interacting protein (RIP), which in turn activates NF-kappaB-inducing kinase and phosphorylates IkappaB. Therefore, the inhibition of IkappaB degradation blocks this RIP-mediated antiapoptosis signaling event. Last, p53 protein levels, and susceptibility to apoptosis, can be deregulated by the human homolog Hdm2 (Mdm2) E3 ligase. This process is inhibited by p53 phosphorylation and by sequestration of Mdm2 by ARF. Better mechanisms to inhibit the ubiquitin-proteasome pathway targeted at the ubiquitin-proteasome

degradation process itself, or more specifically at the E3 ligases known to modulate and downregulate proapoptosis pathways will lead to the enhancement of TRAIL apoptosis signaling and better cancer therapeutic outcomes act through this pathway.

...ABSTRACT: c and second mitochondria-derived activator of caspase (SMAC). This results in the activation of 1caspase - **9** and formation of the **apoptosome** and efficient apoptosis signaling. A third mechanism of the regulation of TRAIL signaling in the...

...of these caspases. IAP binding to caspases can be inhibited by SMAC, which exhibits a **caspase - 9** homology domain. The fourth mechanism for apoptosis activation by proteasome inhibitors is through the stabilization...
...REGISTRY NUMBERS: **caspase - 9** ;
DESCRIPTORS:
ORGANISMS: PARTS ETC: **apoptosome** --
CHEMICALS & BIOCHEMICALS: ... **caspase - 9** --
MISCELLANEOUS TERMS: ...Literature **Review**

6/3,K,AB/7 (Item 3 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)

0014724301 BIOSIS NO.: 200400093070
Chemical-induced apoptosis: Formation of the Apaf-1 apoptosome .
AUTHOR: Cain Kelvin (Reprint)
AUTHOR ADDRESS: MRC Toxicology Unit, University of Leicester, Lancaster Road, Hodgkin Building, P.O. Box 138, Leicester, Leicestershire, LE1 9HN, UK**UK
AUTHOR E-MAIL ADDRESS: kc5@le.ac.uk
JOURNAL: Drug Metabolism Reviews 35 (4): p337-363 November 2003 2003
MEDIUM: print
ISSN: 0360-2532 _(ISSN print)
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Citation
LANGUAGE: English

Chemical-induced apoptosis: Formation of the Apaf-1 apoptosome .
...REGISTRY NUMBERS: **caspase - 9** ;
DESCRIPTORS:
CHEMICALS & BIOCHEMICALS: ...Apaf-1 **apoptosome** complex...

... **caspase - 9** ;
MISCELLANEOUS TERMS: ...Literature **Review**

6/3,K,AB/8 (Item 4 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)

0014582368 BIOSIS NO.: 200300538558
Apoptosis - the p53 network.
AUTHOR: Haupt Susan; Berger Michael; Goldberg Zehavit; Haupt Ygal (Reprint)
AUTHOR ADDRESS: Lautenberg Center for General and Tumor Immunology, The Hebrew University Hadassah Medical School, Jerusalem, 91120, Israel**

Israel
AUTHOR E-MAIL ADDRESS: haupt@md.huji.ac.il
JOURNAL: Journal of Cell Science 116 (20): p4077-4085 October 15, 2003 2003
MEDIUM: print
ISSN: 0021-9533 _(ISSN print)
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Exposure to cellular stress can trigger the p53 tumor suppressor, a sequence-specific transcription factor, to induce cell growth arrest or apoptosis. The choice between these cellular responses is influenced by many factors, including the type of cell and stress, and the action of p53 co-activators. p53 stimulates a wide network of signals that act through two major apoptotic pathways. The extrinsic, death receptor pathway triggers the activation of a caspase cascade, and the intrinsic, mitochondrial pathway shifts the balance in the Bcl-2 family towards the pro-apoptotic members, promoting the formation of the **apoptosome** , and consequently caspase-mediated apoptosis. The impact of these two apoptotic pathways may be enhanced when they converge through Bid, which is a p53 target. The majority of these apoptotic effects are mediated through the induction of specific apoptotic target genes. However, p53 can also promote apoptosis by a transcription-independent mechanism under certain conditions. Thus, a multitude of mechanisms are employed by p53 to ensure efficient induction of apoptosis in a stage-, tissue- and stress-signal-specific manner. Manipulation of the apoptotic functions of p53 constitutes an attractive target for cancer therapy.

...ABSTRACT: in the Bcl-2 family towards the pro-apoptotic members, promoting the formation of the **apoptosome** , and consequently caspase-mediated apoptosis. The impact of these two apoptotic pathways may be enhanced...
...REGISTRY NUMBERS: **caspase - 9**
DESCRIPTORS:
CHEMICALS & BIOCHEMICALS: ... **apoptosome** ; ...

... **caspase - 9** ;
MISCELLANEOUS TERMS: ...Literature **Review**

6/3,K,AB/9 (Item 5 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)

0014555678 BIOSIS NO.: 200300511041
Ways of dying: Multiple pathways to apoptosis.
AUTHOR: Adams Jerry M (Reprint)
AUTHOR ADDRESS: The Walter and Eliza Hall Institute of Medical Research, Melbourne, Victoria, 3050, Australia**Australia
AUTHOR E-MAIL ADDRESS: adams@wehi.edu.au
JOURNAL: Genes & Development 17 (20): p2481-2495 October 15, 2003 2003
MEDIUM: print
ISSN: 0890-9369
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Citation
LANGUAGE: English

...REGISTRY NUMBERS: **caspase - 9**
DESCRIPTORS:
CHEMICALS & BIOCHEMICALS: ... **apoptosome** --...

... **caspase - 9** ;
MISCELLANEOUS TERMS: ...Literature **Review**

6/3,K,AB/10 (Item 6 from file: 55)

0014318601 BIOSIS NO.: 200300273134
Signaling of cell death and cell survival following focal cerebral ischemia: Life and death struggle in the penumbra.
AUTHOR: Ferrer Isidro (Reprint); Planas Anna Maria
AUTHOR ADDRESS: Institut de Neuropatologia, Servei d'Anatomia Patologica, Hospital Princeps d'Espanya, Hospitalet de Llobregat, Universitat de Barcelona, carrer Feixa LLarga sn, Campus de Bellvitge, 08907, Barcelona, Spain**Spain
AUTHOR E-MAIL ADDRESS: 8082ifa@comb.es
JOURNAL: Journal of Neuropathology and Experimental Neurology 62 (4): p 329-339 April 2003 2003
MEDIUM: print
ISSN: 0022-3069 _(ISSN print)
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Focal ischemia by middle cerebral artery occlusion (MCAO) results in necrosis at the infarct core and activation of complex signal pathways for cell death and cell survival in the penumbra. Recent studies have shown activation of the extrinsic and intrinsic pathways of caspase-mediated cell death, as well as activation of the caspase-independent signaling pathway of apoptosis in several paradigms of focal cerebral ischemia by transient MCAO to adult rats and mice. The extrinsic pathway (cell-death receptor pathway) is initiated by activation of the Fas receptor after binding to the Fas ligand (Fas-L); increased Fas and Fas-L expression has been shown following focal ischemia. Moreover, focal ischemia is greatly reduced in mice expressing mutated (nonfunctional) Fas. Increased expression of caspase-1, -3, -8, and -9, and of cleaved caspase-8, has been observed in the penumbra. Activation of the intrinsic (mitochondrial) pathway following focal ischemia is triggered by Bax translocation to and competition with Bcl-2 and other members of the Bcl-2 family in the mitochondria membrane that is followed by cytochrome c release to the cytosol. Bcl-2 over-expression reduces infarct size. Cytochrome c binds to Apaf-1 and dATP and recruits and cleaves pro- **caspase - 9** in the **apoptosome** . Both caspase-8 and **caspase - 9** activate caspase-3, among other caspases, which in turn cleave several crucial substrates, including the DNA-repairing enzyme poly(ADP-ribose) polymerase (PARP), into fragments of 89 and 28 kDa. Inhibition of caspase-3 reduces the infarct size, further supporting caspase-3 activation following transient MCAO. In addition, caspase-8 cleaves Bid, the truncated form of which has the capacity to translocate to the mitochondria and induce cytochrome c release. The volume of brain infarct is greatly reduced in Bid-deficient mice, thus indicating activation of the mitochondrial pathway by cell-death receptors following focal ischemia. Recent studies have shown the mitochondrial release of other factors; Smac/DIABLO (Smac: second mitochondrial activator of

caspases; DIABLO: direct IAP binding protein with low pI) binds to and neutralizes the effects of the X-linked inhibitor of apoptosis (XIAP). Finally, apoptosis-inducing factor (AIF) translocates to the mitochondria and the nucleus following focal ischemia and produces peripheral chromatin condensation and large-scale DNA strands, thus leading to the caspase-independent cell death pathway of apoptosis. Delineation of the pro-apoptotic and pro-survival signals in the penumbra may not only increase understanding of the process but also help to rationalize strategies geared to reducing brain damage targeted at the periphery of the infarct core.

...ABSTRACT: infarct size. Cytochrome c binds to Apaf-1 and dATP and recruits and cleaves pro- **caspase - 9** in the **apoptosome** . Both caspase-8 and **caspase - 9** activate caspase-3, among other caspases, which in turn cleave several crucial substrates, including the...
DESCRIPTORS:
MISCELLANEOUS TERMS: ...Literature **Review**
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$0.88 4 Type(s) in Format 4 (UDF)
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$1.60 TELNET
$67.17 Estimated cost this search
$67.43 Estimated total session cost 5.747 DialUnits

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